THE SYMPTOM-COMPLEX OF OCCLUSION OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY: TWO CASES WITH NECROPSY*

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The symptoms of occlusion of the posterior inferior cerebellar artery are so pronounced and usually so sharply defined, that the clinical diagnosis should be easy, although it may be difficult to exclude implication of the vertebral artery. Three American investigators have made a careful study of obstruction of this artery, but with the exception of these papers by Hun and Van Gieson, and H. M. Thomas, I have been unable to find any thorough consideration of the subject in the American or English literature. It is possible I may have overlooked some paper containing investigation in this line.

The symptom-complex of occlusion of the posterior inferior cerebellar artery is as follows: Sudden onset, usually without disturbance of consciousness. Motor power in the limbs and fifth nerve distribution not affected, or at most temporary and slight weakness of the limbs on the side opposite the lesion. Diminution or loss of pain and temperature sensations in the limbs of the side opposite the lesion, and in the fifth nerve distribution on the side of the lesion, or also in the side of the face opposite the lesion; the disturbance of sensation in the face may be only in the first, or first and second branches, or in the whole distribution of the nerve, depending on the upper level of the lesion. Spontaneous pain, or some form of paresthesia in the area of disturbed objective sensation. Tactile sensation usually intact in all parts. Sense of position usually intact but sometimes affected. Occasionally a zone of intact sensation in the neck between the zone of disturbed sensation in the limbs of one side and the face of the opposite side. Ataxia in the limbs on the side of the lesion (involvement of restiform body, cerebello-olivary fibers), indicating that the fibers of coördination do not decussate below the medulla oblongata. Tendency to fall toward the side of the lesion; tendency for the head to drop toward the side of the lesion. Nystagmus bilateral, more intense when the eyes are directed toward the side of the lesion (Deiters' nucleus (?)). Ménière's symptom-complex; vertigo, revolving vertigo, auditory hallucinations, vomiting (Deiters' nucleus, vestibular nerve and nucleus, cochlear and vagus nerves). Intense headache occasionally. Disturbance of micturition occasionally. Paralysis of the muscles of deglutition on the side of the lesion (vagus) but causing complete inability to swallow, with impaired sensation of the pharynx. Paralysis of the soft palate on the side of the lesion. Paralysis of the larynx on the side of the lesion, voice hoarse, and speaking is in a whisper (loss of phonation with preservation of articulation). Tongue possibly a little weak on the side of the lesion, but the weakness usually not persistent, as the hypoglossus nerve and nucleus usually escape. Taste lost in anterior two thirds of tongue occasionally. Paresis of the sixth nerve on the side of the lesion occasionally. Paresis of the facial nerve on the side of the lesion occasionally. The paresis of the sixth and seventh nerves is often absent in a lesion confined to the posterior inferior cerebellar artery, and possibly it may be caused by further extension of the area of softening. Sympathetic disturbance; smallness of pupil, narrowing of palpebral fissure, retraction of eyeball, on the side of the lesion. Loss of sweating in the face on the side of lesion occasionally, or increase of sweating in the face on the side opposite the lesion, occasionally. Obstinate hiccough. Hemiasynergy on the side of the lesion (Babinski and Nageotte). Pulse may be rapid from paralysis of the vagus. Loss of tendon reflexes, including the patellar tendon reflex, or in other cases exaggeration of the tendon reflexes. Disturbance of hearing.

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With few exceptions I have confined my citation of cases in the literature to those in which a necropsy was obtained.

Dumenil's[1] case 8 reads like one of occlusion of the posterior inferior cerebellar artery, but from the symptoms he describes it is hard to understand his statement that the area of softening certainly was confined to the restiform body.

Leyden's[2] case 1 might have resulted from thrombosis of the posterior inferior cerebellar artery, but he supposed the cause to be embolism of a small artery, and yet the embolus was not found. The lesion was a small myelitic focus in the right half of the medulla oblongata. Objective disturbance of sensation was not detected. The patient had pain in the forehead on the side of the lesion. Leyden's case 2 presented a myelitic focus nearly in the middle of the medulla oblongata. It was supposed to be from senile softening.

Senator's[3] patient had vertigo, but was too ataxic to stand or walk, though he could move all his limbs, and was obliged to go on "all fours," and had a tendency to fall to the left. He had a sensation of cold in the left side of the face, and speech was disturbed as if by some obstruction in the throat or larynx. The left eye was smaller than the right. Every attempt to move the patient caused vomiting. He had temporary diplopia, also difficulty in swallowing. The pulse was 120. Sensation was much disturbed in the left side of the face and in the right limbs and right side of the trunk. Tactile sensation also was involved. The patellar reflex was lost on each side.

The necropsy showed thrombosis of the left vertebral and posterior inferior cerebellar arteries, and softening in the outer and posterior lower part of the left half of the medulla oblongata.

Eisenlohr[4] reports a case which had much the appearance clinically and pathologically of thrombosis of the posterior inferior cerebellar artery, as the lesion corresponded to that caused by occlusion of this artery, and yet he says no large vessel was occluded, and he regarded the lesion as subacute encephalitis.

In Senator's[5] second case the lesion was left-sided and extended from the lower end of the abducens nucleus not quite to the lower end of the hypoglossus nucleus. The left vertebral artery was thrombotic, and the thrombosis extended 2 to 3 mm. on the pons in the basilar artery. The attack began with vertigo. The patient had paresthesia in the right limbs and the left side of the face. Pain sensation was lost in the face only in the distribution of the second branch of the fifth nerve. Associated ocular movements to the left were lost, and sensation for touch pain, temperature, position and passive movement, and motor power, disappeared in the right extremities after a second attack. The case was therefore a complicated one from partial thrombosis of the basilar artery.

In Van Oordt's[6] case the lesion was on the left side and at the level of the middle third of the lower olive, and did not extend into the pons. Hemianesthesia was present on the entire right side of the body, limbs and head, but was not complete everywhere. Tactile sensation on the right side was only slightly affected, and sense of location was intact. Sensation was normal on the left side. The lesion was thrombosis of the posterior inferior cerebellar artery.

Reinhold,[7] case 2: A man had suddenly some disturbance of sensation in the left upper limb, a few hours later complete hemiplegia of the left limbs developed without any disturbance of consciousness. The face was not paralyzed. Nystagmus was present in looking to either side, but more intense in looking to the right. Sensation was much diminished in the left side of the body. The movements of the right upper limb were ataxic. There was complete inability to swallow and right-sided laryngeal palsy. The right vertebral artery was thrombotic from the point of union with the left vertebral artery to a distance of about 3 cm. The basilar artery was not thrombotic. The lesion extended from about the lower end of the inferior olive to the upper part of the hypoglossal nucleus, and almost the entire right half of the medulla oblongata was implicated, including the right pyramid. Much mental excitement was present from the beginning of the paralysis, and the patient was unable to sleep even with narcotics. Reinhold believed this could be explained by interference with the circulation of the anterior part of the brain by the thrombosis of the vertebral artery. Inasmuch as the patient was a physician, and doubtless realized fully the gravity of his condition, it may be that the explanation given is not necessary. Although swallowing was impossible, only one side of the pharynx was paralyzed.

In Wallenberg's[8] case the lesion was on the left side, and was occlusion of the left posterior inferior cerebellar artery and frontal part of the left vertebral artery. Vertigo, and pain in the left eye occurring in the beginning of the symptom-complex, and pain later implicating the entire left side of the face, are explained by irritation of the restiform body, vestibular nucleus, Deiters' nucleus, and the spinal root of the fifth nerve. Slowing of pulse was observed. Diminution of pain and temperature sensations was noted in the right half of the face, and there was also disturbance of the sensations of pain and temperature in the first and second branches of the fifth nerve in the left side of the face. The tactile sensation was not affected. Complete paralysis of deglutition was present. Herpes developed in the areas of disturbed sensation.

The manner in which the symptom-complex of occlusion of the posterior inferior cerebellar artery develops is remarkable. One might well suppose that the destruction of so large an area in the medulla oblongata would be attended by a general disturbance of function. Hun's[9] patient is an example of the manner in which the symptom-complex is likely to develop. The man went to bed feeling fairly well. He awoke during the night with a feeling of a lump in his throat, as if he had swallowed something. In the morning he remained in bed by his wife's advice and not from any feeling of inability to rise. He found he was unable to swallow his breakfast. His voice was hoarse, pulse 80, temperature 99.5°.

In Hun's case there were three areas of softening, but the one which interests us in this connection was in the left side of the medulla oblongata, in the region affected by occlusion of the posterior inferior cerebellar artery. The pathological study was made by Van Gieson. Analgesia and thermic anesthesia were present in the left side of the face and right side of the body. The man was ataxic in the left upper and lower limbs, not in the right. The "muscular sense" was lost only in these limbs. The man showed a tendency to fall toward the left and for the head to fall on the left shoulder, *i. e.*, toward the side of the lesion. This was explained by a lesion of the left restiform body. There was a decided increase in the secretion of sweat on the right side of the face and on the right hand. The fillet was not degenerated, "muscular sense" was lost on the left side, but returned after the first month. Hun attributed the lesion to occlusion of the posterior inferior cerebellar artery.

Ransohoff's[10] case is unsatisfactory, as the clinical history is very deficient. The lesion was on the right side of the medulla oblongata.

In Breuer and Marburg's[11] cases the symptoms were:

Case 1. Right side: hypalgesia on the trunk and limbs. Left side: hypalgesia of face, falling toward the left, ataxia of the upper and lower limbs, disturbance of sense of position in the upper limb, loss of triceps reflex, and sympathetic ophthalmoplegia. Bilateral symptoms were: paralysis of soft palate, paralysis of deglutition.

The left vertebral artery was thrombotic, both posterior inferior cerebellar arteries were intact; therefore the symptoms must be attributed to the occlusion of the vertebral artery and not to occlusion of the posterior inferior cerebellar artery.

Case 2. Right side: mild disturbance of pain sensation on the trunk and limbs (?). Left side: falling toward the left; slight diminution of temperature sensation for cold in the fifth nerve distribution; slight facial paresis; disturbance of hearing; slight weakness of tongue; sympathetic ophthalmoplegia (myosis, ptosis); vasomotor disturbance in the face, and ataxia of limbs. Bilateral symptoms: paralysis of soft palate; paralysis of deglutition; disturbance of senses of position and of passive movement in the upper limbs, slight impairment of lateral movement of the eyes; diplopia, and disturbance of vision.

Thrombosis of the left vertebral and basilar arteries was found following embolism of the vertebral artery.

In the first case the disturbance of pain sensation was in all three branches of the fifth nerve, but was more intense in the third branch. Temperature sensation was unreliable.

The case of Babinski and Nageotte[12] with necropsy was a complicated one. The focus they designate as F.1 corresponds to the area of degeneration caused by occlusion of the posterior inferior cerebellar artery, but three other minute foci were found in the medulla oblongata on the same side. The basilar and left vertebral arteries were thrombotic.

In Mai's[13] case, without necropsy, pain and cold sensations were lost on the left side from the second intercostal space and spine of the scapula downward, and in the right side of the head in the distribution of the first and second divisions of the fifth nerve. The muscles of the throat and larynx were weak, although it seems uncertain whether the larynx was affected. The right palpebral fissure was smaller than the left, and right enophthalmus was present. Sensations of touch, pressure and location were normal everywhere. Sensation of heat was increased in the area of altered sensation. Herpes of the right upper lip was observed. In the affected areas a sensation of warmth was felt.

H. M. Thomas[14] observed two cases of occlusion of the posterior inferior cerebellar artery. In his first case the symptoms were sudden vertigo, pain in the right side of the face, tendency to fall toward the right, vomiting, slight ptosis of right eye, narrowing of right pupil, loss of sweating on the right side of the face, transient lateral nystagmus, some difficulty in swallowing, disturbance of pain and temperature sensations on the right side of the face and in the left limbs and left side of trunk, and ataxia of the right upper limb. The lesion was thrombosis of the right vertebral and posterior inferior cerebellar arteries. There were no marked microscopical lesions. In his second case the symptoms were: recurrent attacks of numbness in the left side of the face, vertigo, difficulty in speech, tendency to fall to the left, dissociated sensory disturbances in the left side of face and right limbs and right side of trunk, paralysis of left vocal cord, loss of sweating on left side of face, diminution of conjunctival, nasal and pharyngeal reflexes on the left side, and slight ataxia in the left upper limb, more marked ataxia in the left lower limb.

It seems strange that in Thomas's first case, in which a necropsy was obtained, no softening was found in the medulla oblongata, and he explains its absence by the rich collateral circulation, but his seems to be the only case in literature of positive occlusion of this artery without softening in the medulla oblongata.

In Müller's[15] first case the symptom-complex developed suddenly during the night. The patient was awakened with vertigo and a feeling that his bed was moving to the right, and with headache soon followed by vomiting. He had paresthesia (sensation of warmth) in the entire left side, including the left side of the face. The cool bed covering felt hot to the left hand, but warm water was cold to the left hand and left side of the face. In urinating he was obliged to wait ten minutes, and he had constipation, and frequent and painful erection during the first few days. He had also dysphagia, and paralysis of the soft palate and larynx on the right side. Sympathetic paralysis was present on the right side of the face; the palpebral fissure and pupil were smaller than those on the left side. The man had a somewhat hoarse and nasal speech. He was not ataxic, but was weak at first in the left side, and after three days the right lower limb became weak for a time from the nearness of the lesion to the pyramidal decussation. The triceps and patellar reflexes were more active on the left side, and there was an indication of the Babinski reflex on this side. Pain and temperature sensations were lost on the left side (diminished in the left side of the neck), but tactile sensation and senses of position and of movement were perfectly normal everywhere on the left side. The sensation in the distribution of the right trigeminus was intact, except that in the distribution of the first branch tactile sensation and pressure sensation were diminished. The pulse was a little rapid.

Müller's second patient awoke one morning with the symptoms, having been well the night before. He was probably syphilitic. The symptom-complex was very similar to that of the first case. Anidrosis was present with the ocular signs of sympathetic paralysis. The facial and hypoglossal nerves were not involved in either case. The pain and temperature sensations in the second case were diminished in the distribution of the first branch of the fifth nerve on the same side as the disturbance of those sensations in the limbs and trunk, but tactile and pressure sensations were intact. The trigeminal nerve of the opposite side was not affected. The corneal reflex was diminished in both cases on the side opposite the disturbance of pain and temperature sensations in the face. In both cases the distribution of the upper branch of the trigeminus was more affected than that of the second and third branches. The mucous membranes of the head in both cases seemed to show no disturbance of sensation. The motor power of the limbs was not affected in the second case, but the patellar and Achilles reflexes were prompter on the side of the sensory disturbances. Ataxia was not present.

The symptom-complex in these two cases was: Dysphagia, paralysis on the side of the lesion of the soft palate, larynx and sympathetic supply, and weakness of the corneal reflex; on the opposite side dissociated sensation involving the entire side, and especially of the first branch of the fifth nerve in the face, and perverse temperature sensation. The absence of ataxia in these cases is noteworthy. Death did not occur in either case. Hiccough is not mentioned. The lesion in one case was evidently left-sided; in the other, right-sided. In the second case glycosuria and albuminuria were present a short time. The intense headache was attributed to the involvement of the fifth nerve. The vesical symptoms in both cases were regarded as bulbar, they were forcible and continued pressing before the stream could be started after the necessity of urination was felt. The painful priapism in one case was noteworthy (both patients were males).

Duret is said to have been the first to state that usually the left vertebral artery is the seat of obstruction, although he had only embolism in mind, but Senator in 1881 expressed the opinion that the left vertebral artery seems more liable to thrombosis, possibly because its course is more in the direction of the subclavian artery, and also because it has a greater blood pressure.

Wallenberg[16] traced the course of the injected posterior inferior cerebellar artery. He found that frequently there is only one posterior inferior cerebellar artery, and when that occurs it is usually the left that is present. Several smaller arteries, branches of the vertebral, take the place of the missing cerebellar artery, but only for the medulla oblongata; and a larger anterior inferior cerebellar artery replaces the missing posterior cerebellar artery. The absence of the right posterior inferior cerebellar artery I have repeatedly noticed.

Wallenberg sums up Duret's investigations on the supply of the vertebral arteries. Each of these arteries, the left the larger, gives origin about 2 cm. below their union to the posterior inferior cerebellar artery, and higher to the anterior spinal artery. Branches from the latter artery enter the raphe and nourish the interolivary bundles, posterior longitudinal bundles, hypoglossus nuclei and other nuclei below the floor of the fourth ventricle. The pyramids are nourished by the anterior spinal arteries and frequently by the vertebral arteries. [It is understandable therefore that when the anterior spinal arteries are not occluded the central and anterior parts of the medulla oblongata are not affected.] Branches from the posterior inferior cerebellar or vertebral artery supply the lateral portion of the medulla oblongata. The posterior inferior cerebellar artery supplies the restiform body. These are all terminal arteries. According to Wallenberg the posterior inferior cerebellar artery is given off 12 to 20 mm. below the union of the vertebral arteries. The free anastomosis of the terminal portion of the posterior inferior cerebellar artery explains the escape of the cerebellum in occlusion of this artery. I have, however, observed an abscess in the terminal distribution of this artery in the cerebellum, without any evidence of implication of the medulla oblongata.

The study of Breuer and Marburg[17] shows that clinically the diagnosis as to whether the vertebral artery or the posterior inferior cerebellar artery is occluded cannot be made with certainty, as the anterior branches of the vertebral artery may supply much the same area (lateral and posterior part of the medulla oblongata) as does the posterior inferior cerebellar artery. Great variations occur in the distribution of the blood vessels. Thus in Breuer and Marburg's first case the vertebral artery was occluded and the posterior inferior cerebellar artery escaped, and yet the lesion occupied about the same region as in those cases in which the latter artery was thrombotic. Breuer and Marburg state therefore that in the majority of cases the diagnosis cannot be made more exactly than that of a lesion of the vertebral artery. When symptoms indicative of pontile lesion occur, the vertebral artery is probably occluded rather than the posterior inferior cerebellar artery. Thrombosis of the vertebral artery, however, may give a different clinical picture from that of thrombosis of the posterior inferior cerebellar artery, in that it presents certain additional symptoms. Especially is this true when the thrombus extends into the basilar artery.

Eisenlohr[18] reported two cases, nos. 3 and 4, in 1879; in one (case 3) the symptoms were suddenly developing right hemiplegia, without loss of consciousness, with disturbance of speech and swallowing, complete paralysis of the tongue eight days later, of the pharynx, complete loss of speech, paresis of the left facial nerve, and partial paralysis of the larynx. In the other (case 4) the symptoms were: Sudden paralysis of the right arm, paresis of the right leg, disturbance of articulation and swallowing, complete loss of speech a few days later, inability to swallow, and paralysis of the larynx.

Eisenlohr mentions that Lichtheim regarded as diagnostic of occlusion of a vertebral artery sudden loss of speech and of the movements of the lips and tongue, paralysis of the larynx and paralysis of the limbs of one side. In Reinhold's case of thrombosis of the vertebral artery hemiplegia occurred.

In my cases of thrombosis of the posterior inferior cerebellar artery, and in most other cases with this lesion, hemiplegia, complete loss of speech, and paralysis of the tongue were not symptoms.

In 1888 Eisenlohr reported the findings in his two cases. In case 3 the vertebral arteries were narrowed and obstructed and numerous branches of the basilar artery entering the pons were obliterated. It seems uncertain whether either vertebral artery was completely thrombotic, but it is so implied. In case 4 the important lesion was old thrombosis of the left vertebral artery with several small foci in the lower half of the pons.

In some of the papers it is distinctly stated that prodromal symptoms preceded the apoplectic attack. Thus Senator's patient felt badly for a few days before the apoplectic insult, and this probably was caused by the beginning thrombosis, or at least, disturbance of the circulation. In the clearly marked clinical picture weakness is not great, but inability to walk may exist because of the marked ataxia (Senator).

The ataxia may be caused by the lesion of the inferior cerebellar peduncle, or of the olivary cerebellar fibers, or of the lower olive, or of the fibers of the vestibular nerve and its nucleus, or of Deiters' nucleus. In some cases ataxia is very striking, thus Senator's patient was obliged to go on all fours, as he could not stand; in my first case also standing at first was impossible, probably largely because of ataxia. The ataxia is usually on the side of the lesion, and may be nearly confined to this side (Reinhold, Hun, Breuer and Marburg, Thomas).

The thrombosis was found on the left side in the cases of Senator (two cases), van Oordt, Wallenberg, Hun, Breuer and Marburg (two cases) and Babinski and Nageotte; and on the right side in the cases of Reinhold, Ransohoff, and Thomas.

The diplopia mentioned in some of the cases is usually temporary, and probably is caused by an interference with the function of the sixth nucleus. My second case shows that at least the lower part of this nucleus may be implicated in the softening. In Senator's second case the lesion extended to the lower part of the nucleus. The abducens nerve was weak on the side of the lesion in Henschen's clinical case.

Weakness of the face occurred in the cases of Breuer and Marburg (case 2), and in my case 2. Degeneration of fibers in the facial nerve explained the facial weakness in the second case of Breuer and Marburg.

Weakness of the tongue was observed in Breuer and Marburg's case 2.

Difficulty in swallowing probably exists in every case, and probably is caused by implication of the nucleus ambiguus. The paralysis is probably unilateral, but the loss of function is complete (Reinhold, van Oordt, Wallenberg, Spiller, two cases). It was complete in a case of unilateral fracture of the base of the skull with paralysis of the vagus on only one side reported by me.[19] The patient was entirely unable to swallow.

Vomiting in some cases is very distressing. Every attempt to move Senator's patient produced it, and in my case 1 it was very intense and could not be controlled.

Slowing of the pulse was observed by Wallenberg, and was attributed by him to irritation of the vagus nucleus.

I have been unable to find any satisfactory explanation for the loss of the tendon reflexes in some cases, and the exaggeration in others; but this variation in these reflexes occurs also in cases of cerebellar tumor. The patellar reflexes were lost in the cases of Senator, van Oordt (except feebly present on reinforcement) and Spiller. In Hun's case the patellar reflex was normal on the right side and a little exaggerated on the left side. Rossolimo[20] mentions diminution of the patellar reflex and of other tendon reflexes, unilateral or bilateral, in five cases of lesions of the brain stem.

The disturbance of the sympathetic fibers in the face on the side of the lesion is very common, and consists of narrowing of the palpebral fissure, narrowing of the pupil, and retraction of the eyeball. Disturbance of sweat secretion probably belongs to this symptom group. All these symptoms are not present in every case. They occur usually on the side of the lesion, and therefore demonstrate that the sympathetic fibers entering the lower part of the cervical cord do not decussate below the upper part of the medulla oblongata. In Hun's case sweating was increased in the face and hand opposite to the lesion. Vasomotor symptoms in the face on the side of the lesion were observed by Breuer and Marburg in their second case. Ptosis also has been observed.

In both cases of Breuer and Marburg the left side of the soft palate was more affected, but both sides were weak. In Hun's case and in van Oordt's the paralysis of the soft palate was bilateral, and this is explained by Breuer and Marburg as the result of bilateral innervation from each nucleus. The nucleus ambiguus, they believe, controls the muscles of deglutition, the larynx and the soft palate.

Disturbance of speech has occurred in most of the cases. It is explained by the implication of the nucleus ambiguus, as this nucleus usually forms the center of the lesion.

The nucleus ambiguus is probably the center for the laryngeal muscles. Van Gehuchten and de Beule believed that it had no connection with the larynx, inasmuch as they found degeneration of the dorsal nucleus after section of the laryngeal nerves. Kohnstamm and Wolfstein[21] have cut the recurrent laryngeal nerve on one or both sides in rabbits and dogs, and have always found the dorsal nucleus intact, but the nucleus ambiguus on the side of the division was degenerated. They therefore regard Van Gehuchten's views as incorrect.

Vertigo is not uncommon (Senator, van Oordt, Thomas, Müller). In Henschen's case the symptoms were those of Ménière's disease—revolving vertigo, hallucinations of hearing, and vomiting—and were explained as a result of lesion of the vestibular nerve or of Deiters' nucleus, cochlear nerve, and vagus.

Subjective sensory disturbances are common. Senator's first patient had a sensation of cold in the face on the side of the lesion; his second patient complained of paresthesia in the limbs opposite to the lesion, and in the face on the side of the lesion. Wallenberg's patient had pain in the eye and entire side of the face, on the side of the lesion. In Hun's patient tingling was felt spontaneously in the face on the side of the lesion, and in the limbs opposite to the lesion following slight friction, *i. e.*, in the analgesic regions, and was evidently a symptom of irritation. Pain was complained of in the face on the side of the lesion in one of Thomas's two cases.

Deep sensation was lost in Hun's case on the side of the lesion, about one month and then returned. The fillet was not found degenerated at necropsy, but the transitory disturbance of the deep sensation may have been due to transitory implication of the fibers forming the fillet.

Sense of position was disturbed in the upper limb on the side of the lesion in one case of Breuer and Marburg, and this they explain by the involvement of the fibers from the nuclei of the posterior columns, especially those from Burdach's nucleus, as these are more exterior.

Tactile sensation usually escapes, but temperature and pain sensations are likely to be diminished, or lost, usually in the face on the side of the lesion, and in the limbs and trunk opposite to the lesion (Senator, Hun, Breuer and Marburg). Tactile sensation may in some instances be impaired, and was so in the cases of Senator. It is possible that the explanation for tactile anesthesia is to be found in implication of the fillet, as some investigators believe that this fasciculus contains tactile fibers.

Hemianesthesia, partial in places, was present in the entire side, including the face, opposite to the lesion, in van Oordt's case; the tactile sensation was only slightly affected. The statement he makes is " Hemianesthesia vom Scheitel bis zur Sohle."

In Wallenberg's case the diminution of pain and temperature sensations in the face were on the side opposite to the lesion, but only for a few days, and this was attributed by him to implication of the central tract of the fifth nerve after the fibers had decussated. In this case pain and temperature sensations were diminished also in the face on the side of the lesion in the first and second branches of the fifth nerve, and this was explained by the involvement of the spinal root of the fifth nerve on the side of the lesion.

The central tract of the fifth nerve, according to Wallenberg, is very near the median fillet in the medulla oblongata, and in front of the hypoglossus nucleus. A lesion to involve this tract must extend nearly to the raphe. The tract passes from the substantia gelatinosa dorso-medially, ventral to the hypoglossus nucleus, across the raphe to the opposite side.

The pathology of the spinal cord has shown with certainty, according to Mai, that a lesion of the spinal root causes complete loss of sensation, whereas a lesion of the central tract causes dissociated sensation. This statement, I think, is questionable.

According to Wallenberg and Schlesinger the first branch of the fifth nerve descends lowest in the spinal root. A lesion corresponding to the lowest and middle thirds of the spinal root of this nerve would give disturbance of sensation of the first and second branches of the nerve.

Henschen,[22] in reporting a clinical case with symptoms such as are observed following occlusion of the posterior inferior cerebellar artery, remarks that tactile fibers ascend in the posterior columns and are next the raphe, therefore they usually escape in these bulbar lesions. He says that he has demonstrated by several cases that destruction of the fillet causes a loss of tactile as well as of pain and temperature sensations, therefore the latter fibers enter the fillet more proximally than the medulla oblongata. If ataxia is on one side and disturbance of pain and temperature sensations is on the other, the lesion is more caudal than when the disturbances are on the same side.

Henschen's patient had normal taste on the posterior part of the tongue, but taste was much affected in the anterior two thirds of the tongue. The entire distribution of the left sensory fifth nerve was affected in his case, so that pain and temperature sensations were lost in this area while tactile sensation was preserved.

Analgesia and thermanesthesia in the trunk and limbs of the side opposite the lesion is explained by Müller by the crossing of the spinal tracts for pain and temperature soon after their entrance into the posterior horns, and the ascent of these fibers in the tractus spinotectalis and tractus spinothalamicus.

From his two cases Müller concludes that the substantia gelatinosa must be regarded as a continuation anatomically of the cervical posterior horn, its disturbance must give sensory symptoms of the posterior horn type, *i. e.,* implication of temperature and pain sensations. The central fibers of this trigeminal nucleus must decussate soon, and the crossed tract would be implicated in the lesions of Müller's two cases. His second case showed the Brown-Séquard symptom-complex in the distribution of the fifth nerve; deep sensation and tactile sensation were diminished in the territory of the first branch on the side of the lesion, and pain and temperature sensations were diminished on the opposite side. This indicates, he thinks, that the fibers of deep sensation and tactile sensation in the spinal root of the fifth nerve have their central tract uncrossed in the medulla oblongata. The first branch has been shown to be connected with the distal end of the terminal nucleus, and this Müller explains by the fact that in the lower vertebrates the first branch (the forehead) does not extend forward as far as the second and third branches. In the conus the lowest roots supply the anal region, which in animals is posterior to the limbs.

In case I of Kutner and Kramer the sensation of the neck was not disturbed, and this escape is explained by the gradual crossing of the sensory fibers from this region, so that they were not all caught in the lesion. In occlusion of the posterior inferior cerebellar artery there may therefore be a sensory zone intact between the disturbed area of the face and that of the opposite side of the body.

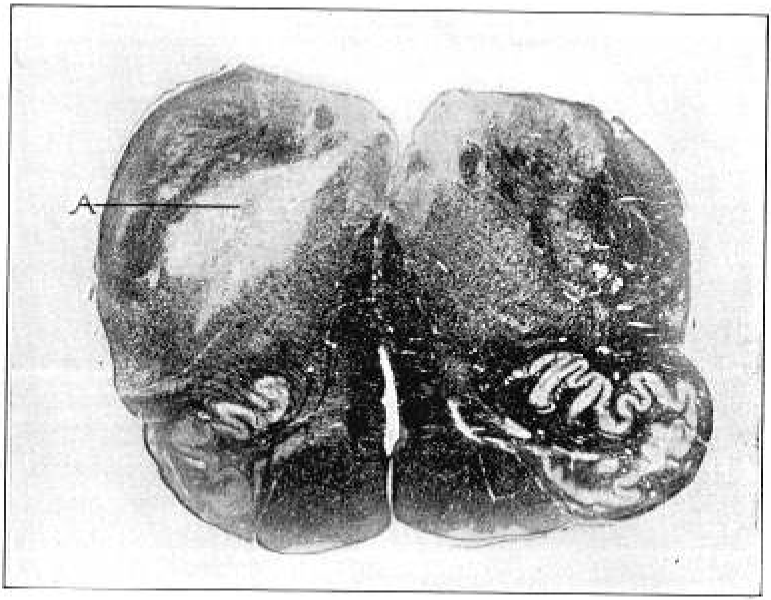

FIG. I. Case I. Showing the lowest level of the lesion A.

My two cases are as follows:

CASE I. Douglas, male, was admitted to the Philadelphia General Hospital, to my service, March 6, 1907. He died March 12, 1907.

Pathological Diagnosis.—Catarrhal and ulcerative enterocolitis, hemorrhagic fibrinous peritonitis; localized chronic adhesive pleurisy, emphysema and edema of lungs; dilatation of right heart; chronic perisplenitis; fatty metamorphosis of liver; cloudy swelling of kidneys; chronic interstitial cystitis.

Notes taken on March 6 are as follows: Chief complaint: Inability to swallow, weakness in left upper and lower limbs. He had a chancre fifteen years ago. Alcohol and tobacco have been used to excess. He has not felt well for three weeks, and stopped work about a week ago, but returned to it again and worked several days. The bowels had not moved for a week; he began last Sunday to take salts after having had his usual cathartic dose on Saturday, and took large doses all day Sunday and during the night. His bowels did not move until Monday morning, when they acted repeatedly, and he had a severe chill. At nine o'clock Monday morning he drank a glass of hot milk and this is the last time he remembers swallowing anything.

At eleven o'clock he tried to get out of bed, but found that he was unable to stand or walk. He slid downstairs to the second floor and called to the family and was carried back to bed. He then found he was unable to drink the milk which was brought him; although he states that he noticed that he was unable to swallow saliva when he first tried to get out of bed. Before he got out of bed he felt dizzy, things swam around, he saw specks before his eyes and then couldn't see at all, although he did not lose consciousness. He had a spasm of the right arm followed by numbness. At present he is unable to swallow anything. He has numbness and some loss of power on the left side. He complains of seeing double and has persistent hiccough. There is no involvement of the bladder or rectum. He is not able to speak above a whisper, although he articulates distinctly. The facial muscles seem to be normal.

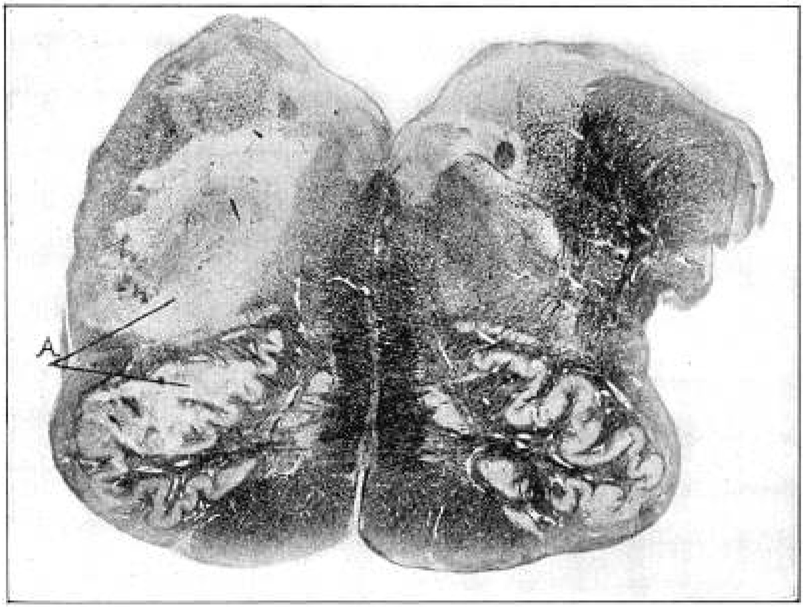

FIG. 2. Case I. Showing the middle level of the lesion *A*.

My examination was made March 8, 1907, and gave the following results: Ptosis of each eyelid is slight and equal on the two sides. He wrinkles the forehead well on each side, closes his eyelids, shows his teeth, draws up well either corner of the mouth separately. The right pupil is a little larger than the left. Reaction to light is present in both eyes but slow. Reaction is prompt in convergence and accommodation. Movements of extraocular muscles are normal. No double vision is noticed at present. The masseter muscle contracts well on each side. In opening the mouth the jaw does not deviate toward the right until the mouth is quite widely open, when the lower jaw goes suddenly to the right, as though there were some defect in the articulation of the lower jaw on the left side. .The tongue may deviate slightly to the right, but this is questionable.

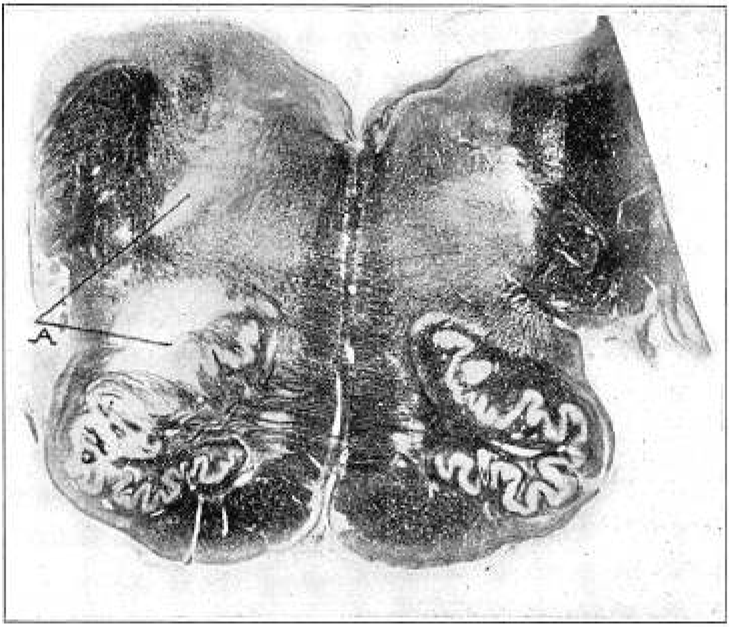

FIG. 3. Case I. Showing the highest level of the lesion *A*.

Sensations of pin prick and touch are normal on the two sides of the face. The soft palate when the man says "ah" is drawn up more on the right side than on the left, this is distinct. He is entirely unable to swallow. The nasal tube is passed without difficulty to a distance of 62 cm. and there seems to be no obstruction to it. He speaks distinctly, but merely in a whisper and expectorates constantly. There is no deafness to the voice and he hears a low ticking watch at a distance of six inches in each ear. The tongue while in the mouth deviates toward the right, more to the right while in the mouth than when it is protruded. The hiccoughing, while constantly present during the observation, has a tendency to occur in attacks of increased severity. The grasp of each hand is good. Biceps tendon reflex and triceps tendon reflex are present, but not very distinct on either side. The movements of the upper limb are free on each side. Sensations of touch and pain are normal in both upper limbs and there seems to be no involvement of either upper limb. Resistance to passive motion is equally good in either upper limb. There is no wasting of the upper limbs. He has little or no tendency to cyanosis of the fingers at present, although Dr. Evans, the resident physician, said the cyanosis was pronounced yesterday morning.

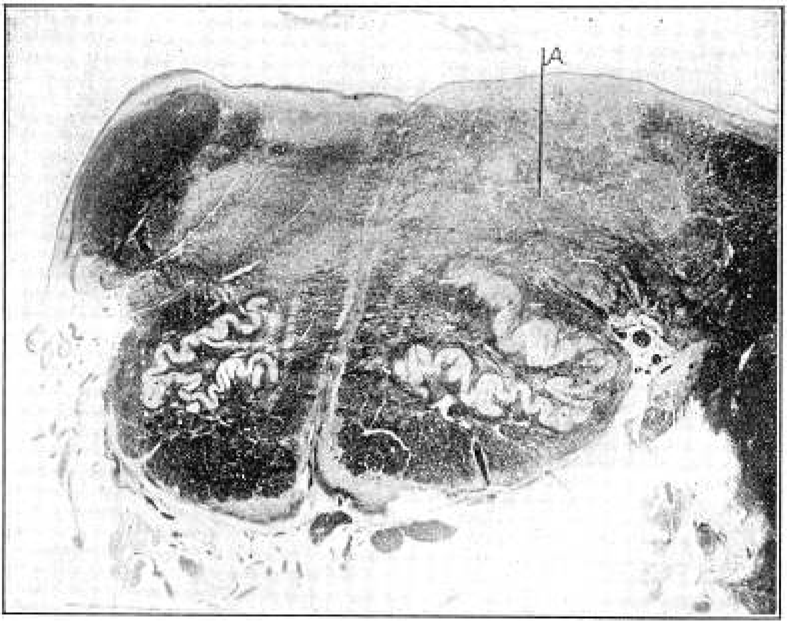

FIG. 4. Case 2. Showing the lesion *A*. The section is reversed in the photograph.

There is no cardiac murmur and no accentuation of the second pulmonic sound. No distinct weakness of the lower limbs is detected. It is very doubtful whether the right lower limb is weaker than the left. The patellar tendon reflex is lost on each side, even on reinforcement. Heel to knee test shows some ataxia on each side. Achilles jerk is lost on each side. Sensations of touch and pain are probably normal in each lower limb, although sensation of pin prick may be somewhat diminished over the right sole. The gait and station are somewhat ataxic. The ataxia is especially pronounced when he is standing with his feet together and eyes closed. He has never had any severe pains in his legs. He says he did not stagger when he walked on the street.

I was unable to see the larynx on account of the epiglottis. Dr. Grayson later made an examination, but the paralysis of the epiglottis prevented him from seeing the vocal cords. He intended to bring suitable instruments and make another attempt, but did not succeed in doing so before the death of the patient.

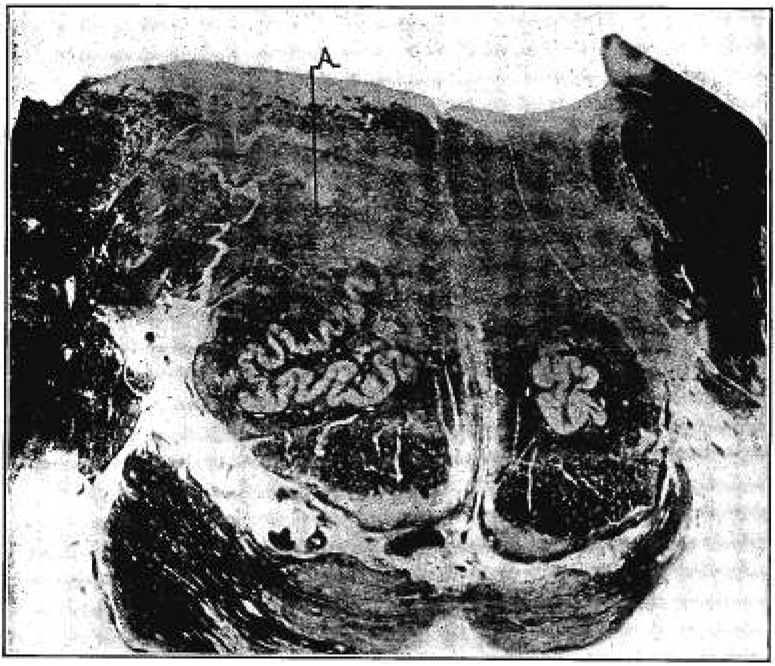

FIG. 5. Case 2. Showing the lesion *A*.

Pulse is 100 at present. Unfortunately the temperature sensation was not tested. Dr. Knipe reported on March 9, 1907: O. D., pupil oval, 4 to 5 mm.; O. S., pupil, 3 mm.; both pupils react faintly to light and convergence; O. D., media clear, fundus negative; O. S., media clear, disc has slightly pale yellowish cast, but no haziness; normal-sized vessels; tension normal.

March 11, 1907: Pulse is 124. Hands and feet are very cyanotic. The vomiting continued last night and this morning; he is weaker. The radial pulse is weak on the right side and is scarcely detectable on the left side. The throat is not very sensitive to manipulation. 11.30 P. M., pulse is imperceptible. He died 1.45 A. M., March 12, 1907. His pulse while he was in the hospital ranged from 80 to 160, and frequently was as high as 120 to 130. Respiration 20 to 30 or 35; temperature on several occasions reached 101 or 102.

The softening with hemorrhagic infiltration is on the left side of the medulla oblongata and extends to the descending root of the glossopharyngeus. It implicates a large part of the restiform body, the anterior part of the descending root of the eighth nerve, a part of the spinal root of the fifth nerve, and the posterior part of the lower olive. It does not extend quite to the periphery of the medulla oblongata, nor does it implicate the lemniscus or nucleus of the hypoglossus. The area of softening in its upper part almost disappears at the level where the restiform body begins to pass into the cerebellum, and in its lower part it is very small at the upper part of the hypoglossus nucleus. The escape of a portion of the sensory root of the fifth nerve, of the fillet, and of the periphery of the medulla oblongata explains the preservation of sensation. The ease with which the nasal tube was passed suggests anesthesia of the pharynx from implication of the vagus and glossopharyngeal nerves.

The diseased area stains a light brown by the Weigert hematoxylin method and contains here and there small recent hemorrhages. The intramedullary portion of the hypoglossus nerve is intact. The nucleus ambiguus is in the degenerated area, and some cells in the position of this nucleus are swollen and greatly degenerated. The anterior pyramids are intact. The nucleus of the hypoglossus on the affected side shows a few degenerated cells, the hypoglossus nucleus of the opposite side is intact. A slight perivascular round cell infiltration is seen here and there in the medulla oblongata. The vessels in front of the pyramids are not notably thickened, but probably are a little so. The pia shows a slight round cell infiltration. Many of the axis cylinders in the necrotic area are much swollen, and this area contains many fatty granular cells.

The left ninth and tenth nerves do not show any degeneration by the Marchi method. No change is detected in a piece of muscle from the left vocal cord, even by the Marchi stain, nor can any change be detected in muscle from the left side of the soft palate by the Marchi stain. It is probable that the duration of life after the lesion occurred was not sufficient for degeneration to be detected even by the Marchi method.

The left vertebral artery is occluded by a thrombus and distended just below where it joins the basilar. The left posterior inferior cerebellar artery shows a partly organized clot.

Case II. Dawson. Male, age 30 years, bartender, was admitted to the Philadelphia General Hospital, to my service, March 27, 1907. He died the same day.

Pathological Diagnosis.—Chronic catarrhal gastritis; active hyperemia of kidneys; edema and congestion of lungs; chronic fibrous pleurisy; right omental hernia.

A history was not obtained from the patient, as he was unable to talk sufficiently, and lived but a few hours after he came to the hospital. He came with an ambulance history of facial paralysis. Duration of illness was one week.

March 27, 1907. Last Monday, three days previous to his admission, just after the patient had arisen in the morning, he felt weak and dizzy. As this feeling passed he found that he was unable to swallow anything, even saliva, and that the left side of the face was very weak; that when he opened his mouth it was drawn strongly to the right. Since then he has been unable to eat or drink and has taken no nourishment for three days.

He has ptosis of the left upper lid. When he shows his teeth his mouth is drawn strongly to the right side. His tongue is well protruded, but deviates apparently to the right, although this may be due to his mouth being drawn so far to the right. His pharynx is anesthetic, as is shown by passing the nasal tube without causing him any annoyance. He is unable to swallow, the fluid passes to a certain distance and then is coughed out. He is able to speak only in a whisper and with much difficulty. His upper and lower limbs on each side are apparently unaffected, as are also the sphincters of the bladder and rectum. His mind is clear, and he is a man of fair intelligence.

The lungs seem clear. He expectorates a considerable amount of grayish mucus. Heart sounds are fairly good. Pulse is 120. Respiration, 40; temperature, 97.

The area of degeneration extends from the level of the upper part of the twelfth nucleus to that of the lower part of the sixth nucleus, and implicates the posterior half of the fillet. A section made at the lower part of the pons where it joins the medulla oblongata shows that a large part of the posterior part of the left side of the section is in recent degeneration. The sixth nucleus is slightly implicated at its lowest portion. The necrotic area stains faintly by the Weigert hematoxylin method. It extends to, but not beyond, the raphe in the pons, and almost to the floor of the fourth ventricle. The restiform body is not implicated, and the spinal root of the fifth nerve only slightly so. The area extends to, but does not invade the lower olive; but implicates the nucleus of the facial nerve. The degenerated area contains numerous small hemorrhages, and is like that seen in the former case, although it is a little higher. The necrotic area contains numerous swollen axis cylinders. The perivascular round cell infiltration is very intense within the medulla oblongata, as is also the round cell infiltration of the pia. The sections present the appearance of syphilis. The basilar and left vertebral arteries are thrombotic, and both are partly occluded by proliferation of the intima. The left vertebral artery is much larger than the right. It is doubtful whether the clot in the basilar artery existed during life. The left posterior inferior cerebellar artery is filled with red blood corpuscles which do not form a distinct thrombus.

The head of the right caudate nucleus is in intense acute degeneration and is filled with fatty granular cells.

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